Amendments to the Specification:

Please amend page 10, the Table beginning on line 10 as follows:

Inhibitor	Description/ comments	Mechanism of C1 inhibition
C1 inhibitor	Plasma serine protease inhibitor	Inhibits C1r and C1s activity
IVIg	Has broad activity	Blocks C1q ligand binding
CRT	Contains several active domains	May inhibit both C1q head and C1q tail
C1Qr	Native C1q receptor	Binds C1q tail, inhibits C1 formation
E. coli C1q binding protein		Binds C1q tail, inhibits C1 formation
gC1qR	Native C1q receptor	Binds C1q head
Decorin	Matrix protein	Binds to C1q head and tail preparations
Chondroitin sulphate proteoglycan	plasma proteoglycan/B cell-secreted	Inhibits C1 formation
Surfactant protein A	Collectin present in the lung	Inhibits C1q ligand binding and C1 formation
HNP-1	Cytotoxic peptide produced by neutrophils	Binds C1q tail and inhibits C1 formation
Peptide gC1q-R_{18} (TDGDKAFVDFLSDEIKEE: SEQ ID NO 1)	Derived from gC1qR	Not defined
Peptide KDIRCKDD (SEQ ID NO. 2)	Derived from CRT	Inhibits C1q ligand binding
Peptide AEAKAKA (SEQ ID NO. 3)	Derived from human IgG	Inhibits C1q ligand binding
Peptide VQVHNAKTKPR (SEQ ID NO. 4)	Derived from human IgG1	Not defined

Table 1 (cont.)

Inhibitor	Description/ comments	Mechanism of C1 inhibition
Peptide WY	Derived from human IgG	Inhibits C1q ligand binding
Peptide 2J (CEGPFGPRHDLTFCW SEQ ID NO. 5)	Synthetic peptide	Binds C1q head, inhibits ligand binding
ghB3	Trimeric C1q B chain	Acts as a competitor for C1q binding
Peptide CBP2 LEQGENVFLQATLL (SEQ ID NO. 6)	Derived from C1q B chain	Acts as a competitor for C1q binding

Please amend page 14, the paragraph beginning on line 12 and ending on page 15, line 6 as follows:

In this connection, peptides directly derived from IgG have been described to inhibit C1q, such as a 7-meric peptide (i.e. AEAKAKA SEQ ID NO. 3) containing the ExKxKx motif, an 11-meric peptide (VQVHNAKTKPR SEQ ID NO. 4) derived from IgG1 that is related to the same motif, and a dimeric peptide (WY, c.f Table 1). These peptides were able to inhibit activation of the classical complement pathway in several *in vitro* assays. However, the WY peptide also inhibits the alternative complement pathway.

Among 42 peptides selected from phage-displayed peptide libraries based on phage binding to human C1q, 20 peptides have been identified, which can inhibit the classical complement pathway in human serum. Remarkably, 13 out of these 20 peptides were able to inhibit the classical pathway as well as the alternative pathway in hemolytic assays, whereas 7 peptides specifically inhibited the classical pathway. Out of these peptides, the peptide 2J (CEGPFGPRHDLTFCW SEQ ID NO. 5) was selected. Peptide 2J is a strong inhibitor of C1q hemolytic function. Similar to the peptides with an IgG motif, peptide 2J binds to the globular head of C1q and inhibits the binding of C1q to IgG. In addition, peptide 2J inhibits C1q from human, primate and rodent origin.

Other selected peptides useful for inhibiting the classical pathway are CEGPFGPRHDLTFCW (SEQ ID NO. 5), CRWDGSWGEVRC (SEQ ID NO. 7), CMWVRMWGDVNC (SEQ ID NO. 8), CFWAGKFGLGTC (SEQ ID NO. 9), CKDRWVVEERCC (SEQ ID NO. 10), and CWNRFKKMDRC (SEQ ID NO. 11). Several other peptides can also be used, which act as a competitor for C1q binding and

are derived from the C1q B chain, e.g. the peptide CBP2 (LEQGENVFLQATLL SEQ ID NO. 6).